Spasticity After Stroke
Its Occurrence and Association With Motor Impairments and Activity Limitations

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Background and Purpose—There is no consensus concerning the number of patients developing spasticity or the relationship between spasticity and disabilities after acute stroke. The aim of the present study was to describe the extent to which spasticity occurs and is associated with disabilities (motor impairments and activity limitations).

Methods—Ninety-five patients with first-ever stroke were examined initially (mean, 5.4 days) and 3 months after stroke with the Modified Ashworth Scale for spasticity; self-reported muscle stiffness; tendon reflexes; Birgitta Lindmark motor performance; Nine Hole Peg Test for manual dexterity; Rivermead Mobility Index; Get-Up and Go test; and Barthel Index.

Results—Of the 95 patients studied, 64 were hemiparetic, 18 were spastic, 6 reported muscle stiffness, and 18 had increased tendon reflexes 3 months after stroke. Patients who were nonspastic (n = 77) had statistically significantly better motor and activity scores than spastic patients (n = 18). However, the correlations between muscle tone and disability scores were low, and severe disabilities were seen in almost the same number of nonspastic as spastic patients.

Conclusions—Although spasticity seems to contribute to disabilities after stroke, spasticity was present in only 19% of the patients investigated 3 months after stroke. Severe disabilities were seen in almost the same number of nonspastic as spastic patients. These findings indicate that the focus on spasticity in stroke rehabilitation is out of step with its clinical importance. Careful and continual evaluation to establish the cause of the patient’s disabilities is essential before a decision is made on the most proper rehabilitation approach. (Stroke. 2004;35:000-000.)

Key Words: motor activity ■ muscle spasticity ■ paresis ■ prevalence ■ stroke

Stroke is characterized by sudden onset of clinical signs related to the site in the brain where the morbid process occurs.1 Damage to the pyramidal tract and its accompanying parapyramidal (corticoreticulospinal) fibers gives rise to the upper motor neuron syndrome,2 including positive and negative features. Positive features include spasticity and abnormal postures, features that are not normally present. Negative features include those that have been lost such as strength and dexterity. Adaptive features, including physiological, mechanical, and functional changes in muscles and other soft tissues, might also develop.3

Initially, some 80% of all patients with stroke experience motor impairments of the contralateral limb(s), ie, hemiparesis.4 In the early literature, abnormal reflexes associated with spasticity were considered to be the major determinant of these motor impairments.5,6 A recent study, conducted in a clinical setting, has reported that 39% of patients with first-ever stroke are spastic after 12 months.7

Spasticity was described by Lance2 in 1980 as “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex.” In 1990, Lance8 reiterated this definition and added that “spasticity does not include impaired voluntary movement and an abnormal posture.” In contradiction to Lance’s definition of spasticity, the tonic stretch reflex has been shown to have a low correlation with the phasic stretch reflex (tendon jerks).9 It has also been recognized that increased muscle tone, ie, increased resistance to passive stretch, is due not only to increased reflex activity but also, and perhaps more, to intrinsic changes of the muscles.3,10 Electromyography (EMG) studies have shown that the reflex-mediated increase in muscle tone reaches its maximum between 1 and 3 months after stroke.9,11 After 3 months, the eventual increased resistance to passive stretch is proposed to be due to intrinsic changes of the muscles.11

Bobath5 and Brunnström,6 pioneers of modern physiotherapy, considered spasticity and hemiparesis after stroke to

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almost always coexist. It has now been recognized that hemiparesis after stroke may occur without spasticity. In an EMG-controlled setting, 24 hemiparetic patients were examined within 13 months after first-ever stroke. One half of the patients had increased resistance to passive stretch associated with muscle contracture, but only in a subgroup of the patients (n = 5) was the resistance to passive stretch linked to neural components (the tonic stretch reflex). Nevertheless, great attention is still paid to rehabilitation techniques based on the assumption that abnormal reflexes are the main purpose of hemiparesis after stroke.

The exact influence of spasticity on motor impairments and activity limitations in stroke patients is difficult to assess because the degree of spasticity may change according to the position of the subject and the task being performed. It has also been suggested that the hypertonicity of leg extensor muscles enables hemiparetic patients to support their body during locomotion.

There is no consensus concerning the number of patients developing spasticity or the actual relationship between spasticity and disabilities after acute stroke. Few studies cover the subject, and as far as we know, no study has been performed 1 to 3 months after stroke, i.e., when eventual spasticity reaches its maximum.

The aim of the present study was to describe to what extent spasticity occurs and is associated with disabilities (motor impairments and activity limitations) initially and 3 months after first-ever stroke.

Subjects and Methods
The patients were consecutively recruited from the Stroke Unit at Danderyd Hospital in the Stockholm area during 10 months (weekends and public holidays not included) in 2001. We included all patients living in Stockholm with an acute first-ever stroke (subarachnoid hemorrhage and cerebellar lesions excluded) with no other risk of falling during gait. One point indicates normal gait; 5 points indicate severely abnormal gait. Inability to walk was also registered. The GUG is considered reliable and valid.

Activities of daily living (ADL) were assessed by the Barthel Index (BI). Patients with <5 points of 100 are considered severely disabled. The BI is considered reliable, valid, and sensitive.

The clinical scales are presented in Table 1.

To avoid subtle side differences without clinical significance, the patients were regarded as hemiparetic only if they fulfilled ≥1 of the following criteria: ≥5-point difference in scores between the affected and nonaffected side of the upper or lower extremities on the BL active movements; ≥2-point difference in scores between the affected and nonaffected side of the upper or lower extremities on the BL rapid movements, ≥5-second difference in scores between the affected and nonaffected hands on the NHPT, and ≥12 points on the SSS item for motor performance.

Ethics
The procedures in the present study were in accordance with the ethical standards of the responsible committee. Patients were given information saying that participation was voluntary and that they could choose not to participate at any time without having to give a reason.

Statistical Analysis
Descriptive statistics were used to present the number of patients with spasticity according to the MAS, self-reported muscle stiffness, hyperreflexia, and clonic beats and to present number of patients with hemiparesis and severe disabilities. Mann-Whitney U test was used for between-group comparisons. Spearman rank-order correlations were used to establish the relationships between muscle tone (spasticity according to the MAS, self-reported muscle stiffness, hyperreflexia, and clonic beats) and the BL, NHPT, RMI, GUG, and BI. Correlation coefficients (positive or, when reversed scales, negative) <0.5 are considered low, those between 0.5 and 0.75 are considered moderate to good, and those ≥0.75 are considered high.
Significance level was set at $P < 0.05$. Data were analyzed by use of Statistica 5.1 for Windows.

Results

Of all 95 patients, 77 (81%) were initially hemiparetic, and 20 (21%) were spastic. Of the 77 hemiparetic patients, 20 (26%) were spastic. Six patients were spastic in both the upper and lower extremity, 13 in the upper extremity only, and 1 in the lower extremity only. The highest estimated MAS scores were 0 ($n = 77$), 1 ($n = 8$), 1+ ($n = 5$), 2 ($n = 4$), 3 ($n = 1$), and 4 ($n = 0$). Among the spastic patients, 12 showed hyperreflexia (all in the upper extremity and 3 also in the lower extremity). Of these, 7 also showed clonic beats, and 6 reported muscle stiffness.

Of all 95 patients, 64 (67%) were hemiparetic 3 months after stroke, and 18 (19%) were spastic. Of the 64 hemiparetic patients, 18 (28%) were spastic. Ten patients were spastic in both the upper and lower extremity, 7 in the upper extremity only, and 1 in the lower extremity only. The highest estimated MAS scores were 0 ($n = 77$), 1 ($n = 8$), 1+ ($n = 5$), 2 ($n = 4$), 3 ($n = 1$), and 4 ($n = 0$). Among the spastic patients, 12 showed hyperreflexia (all in the upper extremity and 3 also in the lower extremity). Of these, 7 also showed clonic beats, and 6 reported muscle stiffness.

The numbers of patients with spasticity, self-reported muscle stiffness, hyperreflexia, and clonic beats are shown in Figure 1. Comparisons between the spastic and nonspastic patients with reference to the motor and activity tests are shown in Table 2.

Correlations between muscle tone and the motor and activity scores were overall low ($r < 0.5, P < 0.05$), except for the initial upper-extremity MAS and BL active movements scores ($r = 0.51, P < 0.001$) and for the 3-month upper-extremity MAS and BL active movements scores ($r = 0.64, P < 0.001$), rapid movements scores ($r = 0.54, P < 0.001$), and NHPT scores ($r = 0.59, P < 0.001$).

### Table 1. Clinical Scales: Scale Value Range and Normal Score

<table>
<thead>
<tr>
<th>Clinical Scale</th>
<th>Scale Value Range</th>
<th>Normal Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAS, spasticity</td>
<td>0, 1, 1+, 2, 3, 4</td>
<td>0*</td>
</tr>
<tr>
<td>Self-reported muscle stiffness</td>
<td>Yes/no</td>
<td>No</td>
</tr>
<tr>
<td>Tendon reflexes</td>
<td>Increased/not increased</td>
<td>Not increased</td>
</tr>
<tr>
<td>Clonic beats in plantar flexors</td>
<td>Present/not present</td>
<td>Not present</td>
</tr>
<tr>
<td>BL motor assessment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active movements, upper extremities</td>
<td>0–57 for each side, 19 subtests</td>
<td>57 for each side</td>
</tr>
<tr>
<td>Active movements, lower extremities</td>
<td>0–36 for each side, 12 subtests</td>
<td>36 for each side</td>
</tr>
<tr>
<td>Rapid movements, upper extremities</td>
<td>0–6 for each side, 2 subtests</td>
<td>6 for each side</td>
</tr>
<tr>
<td>Rapid movements, lower extremities</td>
<td>0–6 for each side, 2 subtests</td>
<td>6 for each side</td>
</tr>
<tr>
<td>SSS, unilateral motor performance</td>
<td>0–18 (arm, hand, leg)</td>
<td>18 for affected side</td>
</tr>
<tr>
<td>NHPT, manual dexterity</td>
<td>Time (s)</td>
<td>&lt;5 s side difference†</td>
</tr>
<tr>
<td>RMI, mobility</td>
<td>0–15</td>
<td>15</td>
</tr>
<tr>
<td>GUG, gait (risk of falling)</td>
<td>1–5</td>
<td>1</td>
</tr>
<tr>
<td>BI, ADL</td>
<td>0–100</td>
<td>100</td>
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</tbody>
</table>

*If >0 on the MAS, patient was considered spastic.
†See Subjects and Methods.
Hemiparesis and severe disabilities 3 months after stroke among patients with and without spasticity in the upper and lower extremities, respectively, are shown in Figure 2.

Discussion

In the present study, we focused on the occurrence of spasticity and its association with motor impairments and activity limitations initially and 3 months after first-ever stroke. Of the 95 patients studied, 21% were initially spastic according to the MAS; 3 months after stroke, 19% were spastic. One third of the spastic patients experienced muscle stiffness. Spasticity was more frequent in the upper than the lower extremities. Three months after stroke, the patients who were nonspastic (n=77) had statistically significantly better motor and activity scores than patients who were spastic (n=18). However, the correlation between muscle tone and the motor and activity scores was overall low, and severe motor and activity problems were seen in almost the same number of nonspastic as spastic patients.

Consistent with earlier findings,4 81% of the stroke patients seen in the present study were initially hemiparetic. Three months after stroke, 67% were still hemiparetic, and 19% were spastic. In a recent study from the United Kingdom, 23 of 59 patients (39%) in a clinical setting were spastic 12 months after first-ever stroke.7 Because of the “late” follow-up, not only neural components but also adaptive features such as intrinsic changes of the muscles may have contributed to the number of spastic patients in that study. The relatively low incidence of spasticity among the hemiparetic patients (28%) in the present study was in accordance with those of O’Dwyer and coworkers3 who found EMG-verified spasticity in only 21% of the hemiparetic stroke patients assessed 13 months after stroke.

It is well recognized that spasticity after stroke may interfere with motor and activity performance, cause pain, and lead to secondary complications.31,33 Initially, the limb may be flaccid and then tone is supposed to emerge, followed by increasing spasticity.31 The use of a neurodevelopmental approach, focusing on normalizing tone and movement patterns, is widespread and claims that inhibition of spasticity should result in an improved motor function.34,35 Some studies have reported reduced spasticity and increased activ-

### TABLE 2. Comparison Between Spastic and Nonspastic Patients According to BL Active and Rapid Movements in the Upper and Lower Extremities, NHPT, RMI, GUG, and BI Initially and 3 Months After Stroke

<table>
<thead>
<tr>
<th></th>
<th>UE</th>
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<th>LE</th>
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<tbody>
<tr>
<td>BLa-UE</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Initially</td>
<td>0 (0–12)</td>
<td>56 (33–57)</td>
<td>&lt;0.001</td>
<td>*</td>
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<tr>
<td>At 3 mo</td>
<td>3 (0–30)</td>
<td>57 (55–57)</td>
<td>&lt;0.001</td>
<td>*</td>
</tr>
<tr>
<td>BLa-LE</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Initially</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>2 (0–10)</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>12 (2–31)</td>
</tr>
<tr>
<td>BLr-UE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initially</td>
<td>0 (0–2)</td>
<td>4 (3–4)</td>
<td>&lt;0.001</td>
<td>*</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>0 (0–4)</td>
<td>5 (4–6)</td>
<td>&lt;0.001</td>
<td>*</td>
</tr>
<tr>
<td>BLr-LE</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Initially</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>0 (0–2)</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>0 (0–2)</td>
</tr>
<tr>
<td>NHPT</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Initially</td>
<td>†</td>
<td>†</td>
<td>†</td>
<td>*</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>&gt;60 (&gt;60)</td>
<td>6 (18–3)</td>
<td>&lt;0.001</td>
<td>*</td>
</tr>
<tr>
<td>RMI</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Initially</td>
<td>1 (0–2)</td>
<td>5 (2–13)</td>
<td>&lt;0.001</td>
<td>1 (0–2)</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>2 (1–7)</td>
<td>12.5 (8–14)</td>
<td>&lt;0.001</td>
<td>2 (2–7)</td>
</tr>
<tr>
<td>GUG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initially</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>&gt;5 (&gt;5–3)</td>
<td>2 (2–1)</td>
<td>&lt;0.001</td>
<td>&gt;5 (&gt;5–3)</td>
</tr>
<tr>
<td>BI</td>
<td></td>
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<td></td>
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<tr>
<td>Initially</td>
<td>10 (5–40)</td>
<td>70 (35–100)</td>
<td>&lt;0.001</td>
<td>10 (5–40)</td>
</tr>
<tr>
<td>At 3 mo</td>
<td>35 (25–50)</td>
<td>100 (85–100)</td>
<td>&lt;0.001</td>
<td>37.5 (35–80)</td>
</tr>
</tbody>
</table>

IQR indicates interquartile range; UE, upper extremity; LE, lower extremity; BLa, BL active; and BLr, BL rapid.

*Not applicable.
†Not calculable (n=5 in the spastic group).
ity performance after botulinum toxin injections in stroke patients. However, there is no evidence that suppression of spasticity by either physiotherapy or medication results in parallel improvements in motor function. Controlled outcome studies have also failed to demonstrate the superiority of any treatment approach in stroke rehabilitation.

In the present study, we excluded patients with recurrent stroke or diseases affecting muscle tone. This was to ensure, as far as possible, that the eventual increased resistance to passive stretch reflected an increased tonic stretch reflex resulting from the present stroke rather than from neural or soft tissues changes caused by earlier stroke events or other neurological deficits. Because spasticity after stroke has been shown to reach its peak 1 to 3 months after onset, we chose a 3-month follow-up. We found spastic patients at 3 months who were not initially spastic, as well as patients with normal muscle tone at 3 months who were initially spastic and/or had increased tendon reflexes (Figure 1). Initial transient cerebral edema and circulation disturbances may be reasonable explanations for this latter phenomenon, thus emphasizing the need for continual evaluation of these patients.

The MAS is often used in clinical practice and research to measure spasticity. The MAS measures resistance to passive stretch, ie, both the tonic stretch reflex and possible intrinsic changes of the muscles; thus, it can be criticized for only reflecting muscle tone of a relaxed limb and for not giving information about activated muscles. We also measured the tendon jerk reflexes, ie, the phasic stretch reflex. In accordance with earlier findings, we found that not all patients with an increased resistance to stretch (≥0 on the MAS) showed increased tendon jerks and vice versa. It has also been recognized that the tonic stretch reflex is of greater clinical significance than the phasic stretch reflex. Additionally, we found only 6 patients who experienced muscle stiffness 3 months after stroke, thus emphasizing the low incidence of spasticity/muscle stiffness among the patients in the present study.

In summary, spasticity seems to contribute to motor impairments and activity limitations and may be a severe problem for some patients after stroke. However, most patients (81%) in the present study were nonspastic, and among hemiparetic patients, only 28% were spastic 3 months after stroke. Also, severe motor and activity problems were seen in almost the same number of nonspastic as spastic patients. Our findings support the opinion of O’Dwyer and coworkers that the focus on spasticity in stroke rehabilitation is out of step with its clinical importance. Careful and continual evaluation to establish the causes of a patient’s disabilities is essential before a decision is made on the most proper rehabilitation approach.

Acknowledgments
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